we are to consider every case giving a positive reaction, a potential active case of tuberculosis, when we must assume that the harboring of a live Tubercle Bacillus anywhere in the body means a condition of potential activity, an attitude that we have consistently assumed with regard to the Spirocheta Pallida. In that event our classification of the disease must be based on the presence of a live bacillus in the tissue of the body, and individuals be considered either uninfected or disinfected (both bacteriologically equivalent), progressive or latent. While this would impose an enormous task in the combat against tuberculosis, it would undoubtedly help more to stamp out this plague than our hitherto accepted classification with its false sense of security.

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THE DIFFERENTIATION OF SYPHILITIC AND TUBERCULOUS PULMONARY LESIONS.*

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As congenital pulmonary lues or so-called "White Pneumonia" is usually fatal in infancy or during foetal life, we may confine our discussion to the acquired forms of pulmonary lues. This condition is regarded as rare by most authors. Thus Fowler [1] was able to find only ten authenticated specimens in the London pathological museums. Only twelve were reported out of 2500 autopsies at the Johns Hopkins Hospital (Osler [2]), while not one was found among 13,000 specimens inthe Army Medical Museum as noted by Clayton.[3]

The number of clinical cases reported in medical literature is relatively greater, but as a positive diagnosis during life is by no means easy, even with the aid of modern diagnostic methods, it is possible that in some of the cases reported as lues of the lungs the diagnosis may have been incorrect. On the other hand it is equally possible that many actual cases of pulmonary lues may have been overlooked or mistaken for tuberculosis.

The diagnosis is rendered difficult by the fact that we may have any of the following conditions present: Tuberculosis of the lungs coexisting with either luetic infection or luetic lesions elsewhere in the body; syphilis of the lungs without tuberculosis present; or finally pulmonary lesions caused by both lues and tuberculosis. In other words, granted that in a given case we have evidence of luetic infection as shown by a definite history of syphilitic infection or by the presence of luetic lesions elsewhere in the body, it still remains necessary to determine whether existing changes in the lungs are due to lues, to tuberculosis, or to both lues and tuberculosis. It is only rarely that we are able to fulfill the postulates laid down by Fowler,[4] namely: (a) A complete history and necropsy finding; (b) Evidence of luetic infection; (c) Repeated negative sputum examinations; (d) Presence of luetic lesions elsewhere in the body.

It is hardly necessary to refer here to the principal differential features laid down in standard text books, namely: the gradual onset and slow course in lues as contrasted with tuberculosis, also the fact that in lues the lesions are usually located at the roots of the lungs or at the base; that stenosis of bronchi and trachea and the formation of bronchiectases are more common in lues, while in tuberculosis there is greater tendency for necrosis and cavity formation. The clinical symptoms may be very misleading.

If we are to accept the diagnosis of a number of authors we may find in pulmonary lues a train of symptoms identical with those occurring in pulmonary tuberculosis, namely: cough and expectoration, fever, night sweats, hemoptysis and gastric disturbances. Nor are luetic lesions always limited to the hilus region or to the base of the lungs. Thus in all five of the cases reported by Landis and Lewis,[5] the diagnosis of tuberculosis had been made and the patients had for a time been treated at tuberculosis sanatoria or health resorts. The same was true in Burnham's case [6] and in both of Brown's cases.[7]

For the reason that modern diagnostic methods had not been perfected at that time, the earlier cases reported in literature, those of Collier, 1882, Hiller, 1884, and Conner, referred to by Roussel,[8] have been left out of consideration. As a basis for this inquiry, however, 31 case histories, reported and published during the last four years, have been studied carefully and the results tabulated. These are the cases reported by the following authors: One each by Culver,[9] Clayton,[10] Roussel,[11] Hays,[12] Hoffman,[13] Lindvall,[14] Blinder,[15] Eastman,[16] Burnham,[17] Gullbring;[18] two each by Brown,[19] and Bauch,[20] five by Landis and Lewis,[21] and seven by Wood.[22]

The 31 cases include also three from our material at the Barlow Sanatorium, not reported or

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published previously. The histories of these three cases were as follows:

No. 673.—A woman married, aged 39, gave a history of tuberculosis in both parents. She had typhoid fever ten years ago, pneumonia eight years ago, pelvic cellulitis and pertonitis ten years ago. There was no history of luetic infection in patient or her husband. She had had some pleurisy on the right side and for eight months before admission suffered from severe colds, with sore throat, irritating cough and some expectoration. There was slight dyspnea, occasional hoarseness, slight fever, night sweats, poor appetite and gastric distress. She had lost sixteen pounds in weight. The tonsils had been removed six months before admission. Ulceration of the throat developed a week or so later. As a child, had enlarged glands of the neck, which had been dissected.

Examination of the chest showed dullness, with bronchovesicular breathing, increased voice and a few fine moist rales over both upper lobes. There was also some dullness with fine rales in the right axilla.

Examination of the throat was made by Doctor Hill Hastings one week before admission, who reported as follows: "Pharyngeal wall shows macerated superficial ulceration, especially marked on lateral walls and extending up into the nasopharynx. The surface is pale red, with infiltration of the mucosa. Tonsillar fossa shows healthy healed scars. There is slight ulceration around the Eustachian tubes, also a shallow ulcer on roof of pharynx. The larynx is negative."

Examination of other organs, including blood and urine was negative. Repeated examination of sputum and the secretion from ulcers was negative for tubercle bacilli. Wassermann test made one week after admission, four plus.

On specific treatment and local antisepsis, the pharyngeal ulcer began to heal. Mixed treatment was instituted one month after admission and was followed by complete healing of the ulcer and marked improvement in general condition. The patient gained eleven pounds and has maintained good health for two years since discharge.

Case 710.—A woman, age 36 years, widowed, gave a family history of tuberculosis. There was one still birth, one miscarriage and she had one living child ten years old who was delicate and had snuffles. At birth the child had an eruption and developed ulcers on the legs.

For three years the patient had been getting more and more tired and languid and had lost thirteen pounds in weight. She had had some cough for six months with scanty expectoration. There had been no hemoptysis. She complained of considerable dyspnea, hoarseness and pain in side and back of chest. Had had some night sweats. The appetite was good and digestion unimpaired. Two years ago had an obstruction of the nose due to some growth. An operation for this condition (Gumma?) was followed by loss of septum. No history of any ulceration on mucous membrane of mouth or throat.

Examination of chest. There was dullness with broncho-vesicular breathing, increased vocal resonance and fine moist rales over both upper lobes as far as the third rib and the spine of the scapula. Above the clavicle on the left side there was a small area of bronchial breathing and voice.

Examination of nose showed absence of septum. The mucous membrane was congested and bled easily. Examination of larynx negative.

Examination of other organs, including blood and urine was negative. Repeated sputum examinations were negative for tubercle bacilli. Wassermann examination made two months after admission was four plus.

The temperature up to this time had been typical of tuberculosis, daily subnormal remission and evening elevation to 100. The pulse range was 80-106.

The patient subsequently developed headache, dizziness, pain in back of neck, nausea and vomiting. Some stiffness of neck muscles, exaggerated knee jerks and some inequality of pupil. Kernig and Babinski phenomenon absent.

She was transferred to the County Hospital and unfortunately lost to further observation.

Case 755.—A man, 36 years old, married, gave a history of tuberculosis in the family and had been associated with an individual suffering with tuberculosis for four years. He gave a history of luetic infection in India ten years ago. For three years has been "run down" and suffered from intermittent cough, hoarseness, little or no sputum, some pain in chest, with loss of appetite, gastric disturbances, insomnia and headaches. Had an attack of iritis one year ago and orchitis three years ago.

Examination of chest: Dullness on both sides, more marked on left with roughened bronchovesicular breathing and increased vocal resonance, but no rales. Signs were heard as far as the third rib in front and the spine of scapula behind. There were no signs at the base.

Examination of nose showed perforation of septum. Examination of other organs, including blood and urine, was negative. Repeated sputum examinations were negative for tubercle bacilli. Wassermann four plus.

Patient was discharged before treatment was begun and unfortunately lost to further observation.

The two following case histories are introduced for the purpose of illustrating the clinical features in the presence of both lues and pulmonary tuberculosis, cases in which a positive sputum examination furnished a basis for differential diagnosis:

Case 862.—A man thirty years of age, railroad employee, gave a negative family history. No other illness, but admitted a Neisser infection eight years ago. Denied Lues. He had a cough and expectoration for six months, but no hemoptysis. There was some dyspnea, a little hoarseness, no pleurisy, no chills and no fever. There had been no night sweats. The appetite was good, the digestion unimpaired. There was some impairment of strength. He had lost ten pounds in weight. The general appearance was pale and spare.

Examination of chest showed dullness, bronchovesicular breathing and increased vocal resonance over both upper lobes as far as the second ribs in front and the spine of the scapula behind. A few fine moist rales were heard on both sides and some pleuritic friction rales at the left base.

Examination of nose and throat showed congestion of pharynx extending to soft palate and uvula. Examination of larynx unsatisfactory. Teeth in poor condition. Pyorrhea present.

Temperature during residence occasionally 99, or 99.2, pulse range 65-90. Sputum examination showed typical tubercle bacilli present on direct examination. Other organs, including blood and urine, negative.

About one month after admission developed an ulcer of the tongue. Wassermann examination was four plus.

Patient was discharged before treatment, but it was reported subsequently that the ulcer on the tongue had healed promptly under specific treatment.

Case 831.—Man, mechanician by occupation, 21 years old, single, gave a history of tuberculosis in brothers and sisters and had been exposed to infection at home. Habits good. Denied venereal infection.

For about one year had had cough with some expectoration. The sputum was not examined before admission. Had had several hemoptyses. There was dyspnea on exertion and some pain in the chest. He did not complain of hoarseness, and had had no chills, though there was some fever

SUMMARY OF FINDINGS IN CASES REPORTED.

Author	No. of Cases	Lues Infect. History	Luetic Lesions Other Organs	Location of Pulmonary Lesions	Wassermann Test	T. B. Sputum	Remarks
Culver	1	+	+	Apices	+	0	Resolved
Clayton	1	+	+	Scattered	+	0	Necropsy
Roussel	1	+	+	Scattered	+	0	Resolved
Hays	1	0	0	Base	+	0	Untreated
Hoffman	1	+	+	Apices	+	0	Resolved
Lindvall	1	(?)	—	Hilus	+	0	Resolved
Blinder	1	+	+	Apex and Hilus	+	0	Resolved
Bauch	2	+	0	Hilus	+	0	Compl. Fix. Tbc.
Easton	1	+	0	Base	+	0	———
Burnham	1	+	+	Apex and Base	+	0	Resolved
Wood	7	+	0	Apex and Base	+	0	Resolved
Landis and Lewis	5	+	0	Apices	+	0	Resolved
Gulbring	1	+	+	———	+	0	Necropsy
Downing	2	—	—	Apices	—	0	Resolved
Brown	2	+ (1)	+ (1)	Scattered	+ (1:?)	0	(1) Resolved Necropsy, (1) Aneurism Aorta
Klots	3	+ (1)	+ (2)	Apices (2), Base (1)	+	0	
Total	31	+ (24)	+ (10)	Apices (22)	+(30)	0(31)	

and night sweats at the time of the hemoptyses. His appetite was good, the digestion unimpaired, but he had lost some strength and about eighteen pounds in weight.

Examination of chest: There was dullness with bronchovesicular breathing and increased vocal resonance over both upper lobes as far as the second ribs in front and the fourth dorsal vertebra behind. Fine moist rales heard over right side as far as base in front and the angle of the scapula behind. On the left side a few crackling pleuritic rales in the axilla.

Sputum examination showed typical tubercle bacilli on direct examination. Examination of other organs, including blood and urine, was negative. The temperature on admission was normal and the pulse range 85-110.

About two weeks after admission developed genital ulcers. These were at first mistaken for herpetic vesicles. But subsequently they coalesced and developed into a dirty, punched out ulcer, with some induration. The glands in the left inguinal region became enlarged, slightly tender and matted together.

Microscopic examination of preparation from ulcer made by Dr. Walter Brem was positive for Spirocheta Pallida.

Wassermann test suspicious.

Patient referred elsewhere for treatment and lost to observation.

The accompanying tabulation gives a summary of the different cases reported. Thus we find that only three fulfilled Fowler's postulates. There was, however, a definite history of luetic infection in all but seven, and in one the history of infection was doubtful. The apices or upper lobes alone were involved in eleven, while in thirteen the lesions were located not only at the tops of the lungs but also at the base or hilus region. In only six were the lesions limited to the base or hilus. This distribution of lesions would therefore be contrary to the generally accepted statements in text books.

The Wassermann test was positive in all of the thirty cases in which it was employed. In one case no statement is made in regard to the Wassermann.

Sputum examinations were negative for tubercle bacilli in every one of the cases. Four observers had employed the antiformin method in addition to the direct method. Three had made animal inoculations with sputum, and one observer reports twenty-four successive examinations. One used the complement fixation test. All the above tests were negative for tuberculosis. Three observers reported the results of X-ray examinations.

In twenty-two cases the clinical symptoms and local symptoms cleared up under some form of specific treatment. Three died, while three were not observed after treatment or were not treated. In one case there was no change in condition, and in one other no statement was made as to treatment.

The important clinical features derived from the study of these cases is the fact that every one of them showed symptoms characteristic of pulmonary tuberculosis; the symptom complex present including cough, expectoration, hemoptysis, emaciation, fever, night sweats and gastric disturbances. In all a diagnosis of tuberculosis had been made originally, to be modified or altered later. A number of the patients had been admitted and had been under observation or treatment at tuberculosis institutions or resorts before a diagnosis of lues had been made. The physical signs in the chest as described were almost identical with those of pulmonary tuberculosis. In addition to changes of resonance and breath sounds, such as dullness, impaired resonance, bronchovesicular breathing or bronchial breathing, moist rales or other adventitious sounds were reported in most instances. When it is recalled how difficult it may be for the pathologist to distinguish luetic and tuberculous lesions by anatomical or even histological examination; it can be readily understood that their differentiation during life by clinical methods may be puzzling or difficult. It is necessary to be somewhat guarded, therefore, in accepting a diagnosis in the absence of definite bacteriologic or biologic proof. Nor can we accept as competent evidence necessarily the results of negative sputum examinations. The inconclusiveness of sptum examinations has been pointed out by Cunningham,[23] and has been repeatedly emphasized. The medical reports of numerous tuberculosis sanatoriums show that on an average in only about 50 per cent. of the cases are tubercle bacilli found in the sputum at any time.

Ten of the cases showed luetic lesions elsewhere in the body, but this occurrence is not necessarily a proof that existing lesions in the lung were also luetic. Tuberculosis and lues may be and very frequently are associated. We know that a pre-

vious or recent luetic infection may either predispose to tuberculosis or cause a latent tuberculous process to light up or become active. For that reason it cannot be accepted as a definite proof that in fifteen cases the symptoms and physical signs in the lungs cleared up promptly under specific antiluetic treatment. It may be properly urged that the effect of such treatment in improving the general condition and resistance of the body may indirectly exert a favorable influence upon the tuberculous process.

It is quite usual to find in cases of chronic pulmonary tuberculosis alternating periods of quiescence and activity, such exacerbations being brought about by intercurrent infections or unfavorable circumstances. The possible association of lues and tuberculosis must therefore be kept in mind constantly, bearing as it does on the question of treatment. It is generally accepted that by treating the luetic condition we may accelerate improvement or arrest of a tuberculous infection. Antiluetic treatment would be indicated therefore even if the pulmonary lesions were tuberculous and not luetic. In this connection, however, some warning notes have been sounded as to the danger of causing focal reactions in the lungs by the administration of salvarsan and neosalvarsan. Attention has been called to this question recently by Potter,[24] who recommends their use, however, in latent incipient or chronic fibroid pulmonary tuberculosis.

Recapitulating our present knowledge as to the differentiation of pulmonary lues and tuberculosis, it would appear that a diagnosis must be made cautiously and not until all diagnostic methods at our disposal have been exhausted. The following procedures would suggest themselves: Repeated sputum examinations both by the direct and antiformin methods; cultures with Petroff's medium or some similar method of direct planting; animal inoculation and routine Wassermann tests in all cases with negative sputum examination, where definite lung signs are present. A reliable method of complement fixation for tuberculosis would be of considerable value in this connection, as obviously none of the tuberculin tests possess any practical clinical value. X-ray examinations and fluoroscopy may also throw much valuable light as to conditions present in the chest. Wood[25] recommends that in addition to X-ray examinations of the chest, radiographs be made of the long bones, in cases where lues is suspected. Needless to say a painstaking history and careful general physical examination should always precede the above special examinations.

SUMMARY.

Summing up the results of this clinical study we find that in thirty-one cases reported as pulmonary lues there were present symptoms and physical signs generally accepted as characteristic of pulmonary tuberculosis, and that in the majority of these cases a diagnosis of pulmonary tuberculosis had been made and that many of the patients had been treated as such. A subsequent diagnosis of pulmonary lues had been made only after the usual and accepted methods of diagnosis had been employed and additional facts obtained by careful histories and more thorough examinations. The experience and reputation of many of the authors reporting these cases would in itself assure us of the correctness of the diagnosis as reported. At the same time certain sources of error have been pointed out and conceded, while possible objections have been anticipated.

At a time when many are beginning to recognize certain limitations and sources of error in the diagnosis of pulmonary tuberculosis, it may be well to recall other conditions that may bring about pulmonary changes. We certainly may err in making a diagnosis of tuberculosis on insufficient grounds and in so doing subject our patient to considerable loss, annoyance and worry. The clinical, social and economic significance of such errors is too obvious to require further discussion.

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METABOLISM AND DISEASE.*

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The charge has been made in the past that medicine has not kept pace with surgery. This charge, unfortunately, cannot be denied, and the reason, we are forced to admit, has been in part at least, due to the slow growth in physiology. At the present time, however, the pendulum is swinging the other way. Not only are physiologists producing satisfactory solutions to many heretofore unsolved problems, but clinicians are more interested in acquainting themselves with these results, and there are more means of bridging the way from the clinic to the research laboratory, which, in many places, is now considered a necessary adjunct to the hospital. The illuminating work being done at the present time, on organ functions, ductless glands, blood chemistry, serum ferments, metabolism and nutrition, all point unmistakably to the fact that physiology is coming into its own. The body is recognized not only as an anatomical structure, but more as a living structure, with various closely related interdependent normals, the physiological actions of which are bio-chemical, and the life of the organism depends upon the ever-changing activity of its cells which are never in a state of equilibrium. The supply which the blood is constantly

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